CD47 is upregulated on circulating hematopoietic stem cells and leukemia cells to avoid phagocytosis.

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Scientific Abstract:
Macrophages clear pathogens and damaged or aged cells from the blood stream via phagocytosis. Cell-surface CD47 interacts with its receptor on macrophages, SIRPalpha, to inhibit phagocytosis of normal, healthy cells. We find that mobilizing cytokines and inflammatory stimuli cause CD47 to be transiently upregulated on mouse hematopoietic stem cells (HSCs) and progenitors just prior to and during their migratory phase, and that the level of CD47 on these cells determines the probability that they are engulfed in vivo. CD47 is also constitutively upregulated on mouse and human myeloid leukemias, and overexpression of CD47 on a myeloid leukemia line increases its pathogenicity by allowing it to evade phagocytosis. We conclude that CD47 upregulation is an important mechanism that provides protection to normal HSCs during inflammation-mediated mobilization, and that leukemic progenitors co-opt this ability in order to evade macrophage killing.